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ORIGINAL ARTICLE

Extrahepatic arteries of the human liver – anatomical variants and surgical relevancies

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Summary

The purpose of our study was to investigate the anatomical variations of the extrahepatic arterial structures of the liver with particular attention to rare variations and their potential impact on liver surgery. A total of 50 human abdominal organ complexes were used to prepare corrosion casts. A multicomponent resin mixture was injected into the abdominal aorta. The portal vein was injected with a different colored resin in 16 cases. Digestion of soft tissues was achieved using cc. KOH solution at 60–65 °C. Extrahepatic arterial variations were classified according to Michels. All specimens underwent 3D volumetric CT reconstruction. Normal anatomy was seen in 42% of cases, and variants were seen in the other 58%. No Michels type VI or X variations were present; however, in 18% of cases the extrahepatic arterial anatomy did not fit into Michels' classification. We report four new extrahepatic arterial variations. In contrast to the available data, normal anatomy was found much less frequently, whereas the prevalence of unclassified arterial variations was higher. We detected four previously unknown variations. Our data may contribute to the reduction of complications during surgical and radiological interventions in the upper abdomen.

Introduction

The arterial anatomy of the liver is highly variable with normal anatomy present in 50.7% [1]–80.9% [2] of cases. Variations of the extrahepatic arterial system are of great importance in terms of liver resections, living donor hepatectomies, whole-organ and split liver transplantation from deceased donors. Recognition, and if necessary, appropriate reconstruction of anomalies is essential to minimize perioperative morbidity and mortality related to ischemic parenchymal and biliary complications. Aberrant hepatic arteries can also be of major significance during operations on the gallbladder, pancreas, and the upper intestinal tract. Furthermore, they can present technical challenges during transarterial chemoembolization and infusion therapy of liver tumors.

The first description of the aberrant hepatic arteries was published in 1756 by Haller [3]. Since then the anatomic variations of the extrahepatic arteries have been examined by several authors worldwide. In 1966, Michels' classic series of 200 autopsies [4] defined the basic anatomic variations in hepatic arterial supply and has served as the benchmark for all subsequent contributions in this area, such as the simplified classifications of Hiatt *et al.* [5] and Varotti *et al.* [6].

The aim of our study was to investigate the branching patterns of the celiac trunk and the superior mesenteric artery contributing to the blood supply of the liver, paying particular attention to rare variations not reported in previous studies. Vascular corrosion casting technique and 3D volumetric CT reconstruction were used to precisely delineate the anatomy and anatomic variations.

Materials and methods

Vascular corrosion casts were prepared from a total of 50 abdominal organ complexes obtained from fresh human corpses of Caucasoid race. Written permission was obtained beforehand from the Ethical Commission of Semmelweis University. The corpses neither had any history of liver disease, nor presented any signs of abdominal trauma or macroscopic alteration.

Following the preparation of the abdominal aorta, a polyethylene cannula was inserted into its proximal end. Lumbar branches, renal arteries, and the aorta above the origin of the inferior mesenteric artery were ligated. To begin the investigation of the spatial relationship between the main portal vein and the hilar arteries, the portal vein was also injected in 16 cases.

For leak control, the specimens were flushed with warm tap water through the abdominal aorta (and the portal vein, if cannulated) to detect and eliminate resin outflow further on.

A special vinyl ester resin mixture developed by M. Kiss was prepared for injection. The components of the mixture were as follows: 1. Resin: Novolac-based Epoxy Vinyl Ester Resin (Derekane 470-300 by Ashland); 2. Pigments (5%): FP3000 red and FP1021 yellow (by Cytec Surface Specialties Austria GmbH); 3. Accelerator (2%): Cobalt 2-ethylhexanoate, *N*,*N*-Dimethyl aniline (Accelerator NL-23 by AkzoNobel); 4. Catalyst (2%): Methyl Ethyl Ketone Peroxid (Butanox M-50 by AkzoNobel). The pigment guaranteed not only different colors of the different vessels, but also provided suitable CT density (approx. 250 HU). During the injection, the liver was afloat in tap water at room temperature. The viscosity of the resin was set to enter the arteries with a diameter of 0.3–1 mm. After filling the arteries with resin, the proximal end of the aorta was clamped. The resin polymerized in 3–5 min, after which, concentrated KOH was added to the surrounding water to commence digestion of the soft tissues. The corrosion process lasted 1 week at 60–65 °C. Residual fat and liver parenchyma were removed by rinsing in warm water, leaving only the cast behind.

Classification of the extrahepatic arteries was based on Michels' classic results of 200 autopsies [4]. Aberrant hepatic arteries can be accessory, occurring in addition to the normal arterial pattern and supplying only partially the left or the right lobe; or replaced, representing the primary arterial supply to the lobe. In addition, as our organ complex casts were suitable for investigation of the complete upper abdominal vascular structure, the right gastric artery (RGA), the gastroduodenal artery (GDA), and the interrelationship between the main portal vein and the hilar hepatic arteries were also taken into account due to their high surgical and radiological significance.

CT images of each ex situ organ complex specimen were acquired, anonymized, and interpreted in random order by an experienced radiologist. For CT examinations, a Philips Brilliance 16 multidetector CT (parameters: 140 kV, 300 mAs, collimation: 16 × 0.75 mm, overlap 50%) was used. Specimens were placed in their anatomical orientation. Images with a pixel spacing 0.08 × 0.08 mm and with 0.4 mm axial resolution were obtained, and multiplanar reconstructions were used for image evaluation. Branching systems were demonstrated in 3D volumetric reconstruction.

Results

Arterial variations classifiable by Michels

In our series of 50 corrosion casts, 41 casts (82%) could be classified according to Michels. Twenty-one cases (42%) showed normal arterial pattern (Michels I), while 29 casts (58%) presented different types of extrahepatic arterial variations. However, nine casts (18%) displayed variations not described in the Michels' classification.

Replaced hepatic arteries

Replaced left hepatic artery (r-LHA) arising from the left gastric artery (LGA) – Michels II – was observed in 3 (6%), while replaced right hepatic artery (r-RHA) originating from the superior mesenteric artery (SMA) – Michels III – was present in 7 (14%) cases.

Double replaced system (Michels IV – Fig. 1) was present in two casts (4%).

Accessory hepatic arteries

Accessory left hepatic artery (a-LHA) – Michels V – was present in 4 (8%) cases, accessory left and right hepatic arteries together (a-LHA and a-RHA) – Michels VII – in 1 (2%) case, while combined a-LHA and r-RHA (Michels VIII) were found in 2 (4%) cases. One cast showed a

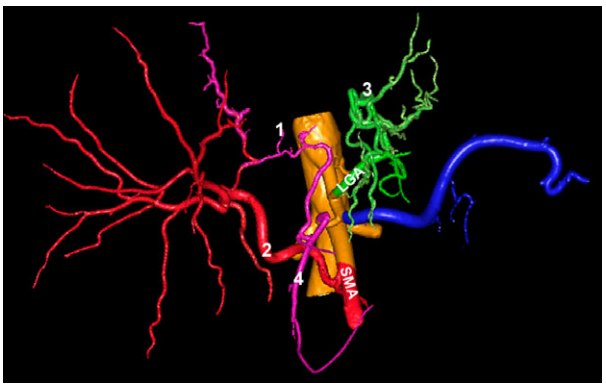

Figure 1 Double replaced system. 1: artery of the caudate lobe (purple); 2: r-RHA (red); 3: r-LHA (green); 4: GDA; Blue: SA. Anterior aspect.

replaced common hepatic artery (r-CHA) arising from the SMA (Michels IX – Fig. 2).

It is notable that in four cases we observed well-defined Michels types showing additional arteries with extrahepatic destination. In the Michels I group, one cast presented right gastric artery (RGA) and a-LGA originating from LHA, another cast showed an a-LGA arising from the artery of segment II (A2) and segment III (A3). In Michels type V, we found one cast having an a-LGA from the a-LHA. The only case of Michels type VII presented two a-LGA arteries branching from LHA, which represents a triple accessory system, a structure which can be considered as a subtype of Michels VII.

No casts presented a single a-RHA from SMA (Michels VI) or r-CHA originating from the left gastric artery (Michels X).

Arterial variants not mentioned in Michels' classification (Unclassified variations – UC)

Of 50 cases, nine corrosion casts (18%) showed unusual arterial patterns that could not be classified according to Michels.

The UC variations of our series could be divided into two groups. The first group consisted of five cases presenting arborization abnormalities of the CHA. Trifurcation of CHA was observed in four cases overall, with the CHA giving off the RHA, LHA, and the gastroduodenal artery (GDA) in three casts. One cast showed an early, proximal origin of the RHA from the CHA, which results in the CHA trifurcating into LHA, GDA, and RGA (Fig. 3a–c). In this newly described variation, the RHA ran behind the portal vein.

The fifth case within this group is also a new variant, the CHA branching into five arteries: the LHA, RHA, artery of

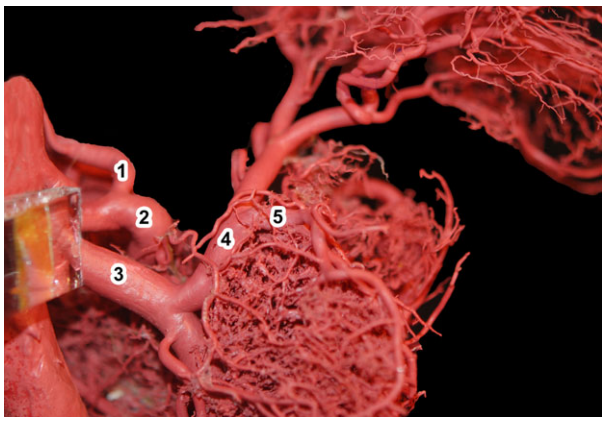

Figure 2 Replaced CHA originating from SMA. 1: LGA; 2: SA; 3: SMA; 4: CHA; 5: GDA. Right dorsolateral aspect.

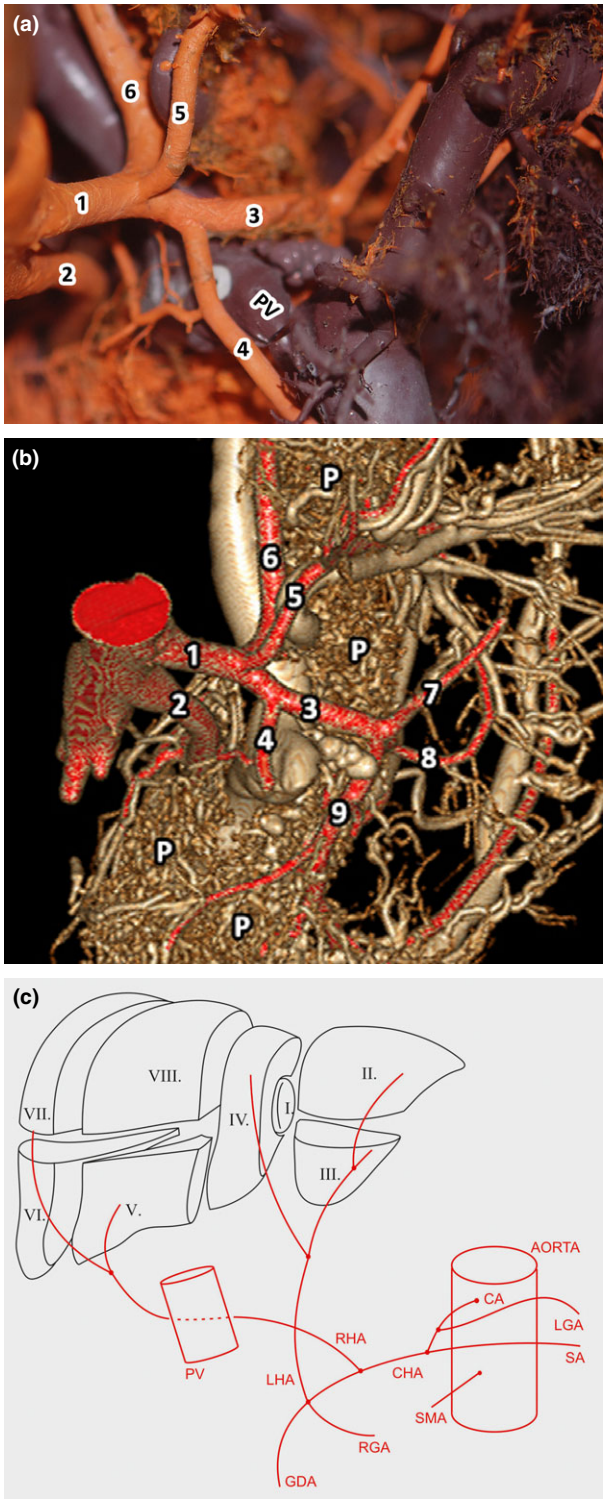

Figure 3 (a–c) Proximal branching of RHA with retroportal course. CHA trifurcation into LHA, GDA, RGA. New UC variation. 1: CA; 2: SMA; 3: CHA; 4: RHA; 5: LGA; 6: SA; P: pancreas; PV: portal vein. (a,b) right lateral, dorsocranial aspect.

segment IV (A4), GDA, and RGA (Fig. 4a–c). It is also remarkable that the caudate artery (artery of segment I–A1) originated dorsally from the RHA only 4 mm away from the point of pentafurcation (Fig. 4a,b).

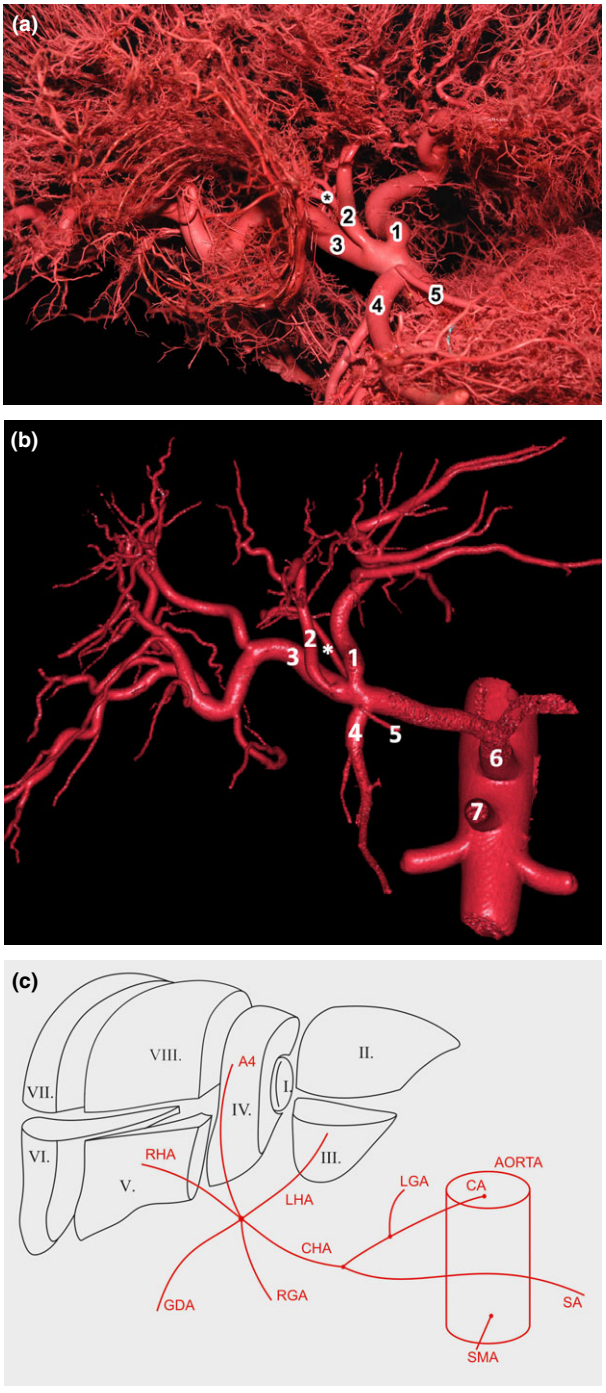

Figure 4 (a–c) Common hepatic artery pentafurcation. New UC variant. 1: LHA; 2: A4; 3: RHA; 4: GDA; 5: RGA; 6: CA; 7: SMA; *: A1. Anterior aspect.

The second group of the UC variations is formed by the four cases displaying anomalous origins and courses of the lobar and sectorial arteries, the RHA, LHA, right posterior hepatic artery (RPHA). In one case, the RHA arose from the celiac axis (CA) and coursed behind the portal vein. In another cast, displaying a new variant, the RHA originated from the proximal part of the CHA and then passed in front of the portal vein. Thus, CHA did not bifurcate into PHA and GDA, but into RHA and LHA-GDA trunk (Fig. 5a–c).

In one cast, exhibiting the fourth new variant, the RPHA arose directly from the CHA, in close proximity to the CA bifurcation. It then passed around the portal vein to reach the right posterior sector of the liver. The CA bifurcated into the splenic artery (SA) and CHA; and the LGA arose independently from the aorta (Fig. 6a–c).

The last, already known UC variation of this group is a proximal branching of the LHA with a considerable distance between the origins of the LHA and RHA. In this case, RHA took off of GDA (Fig. 7).

Thus, among these 9 UC variations, we encountered four cases which, to the best of our knowledge, have not been reported before: CHA pentafurcation; proximal origin of RHA from CHA with retroportal course, CHA trifurcates into LHA, GDA, and RGA; proximal origin of RPHA from CHA with retroportal course, CHA gives the LHA-right anterior hepatic artery (RAHA) common trunk and GDA (LGA originates separately from aorta); proximal origin of RHA from CHA with a course in front of the portal vein, CHA later divides into LHA and GDA.

Vessels of approximately 1 mm diameter were visualized during 3D CT evaluation of the specimens and all variations were recognized. Therefore, radiological and anatomical results were identical.

Discussion

Couinaud in his classical work [7] analyzed arterial vascular casts which were prepared by injection of the arteries at the level of the hepatic pedicle without specifying the source of these arteries except for the left gastric artery that had been checked. Therefore, we designed a study to reveal the hepatic arterial vascular system originating from normal and variant sites. For this purpose, we investigated the vascular structure of abdominal organ complexes instead of liver casts that provide only limited information about the blood supply from extrahepatic arterial source. Moreover, our casts provide 3D data on the whole upper abdominal vascular system making these data equally important for all interventions in this region. Furthermore, the 3D CT reconstructions of these casts simulate the preoperative angiographies. Our series of 50 human liver casts is to our knowledge, the largest sample of its kind.

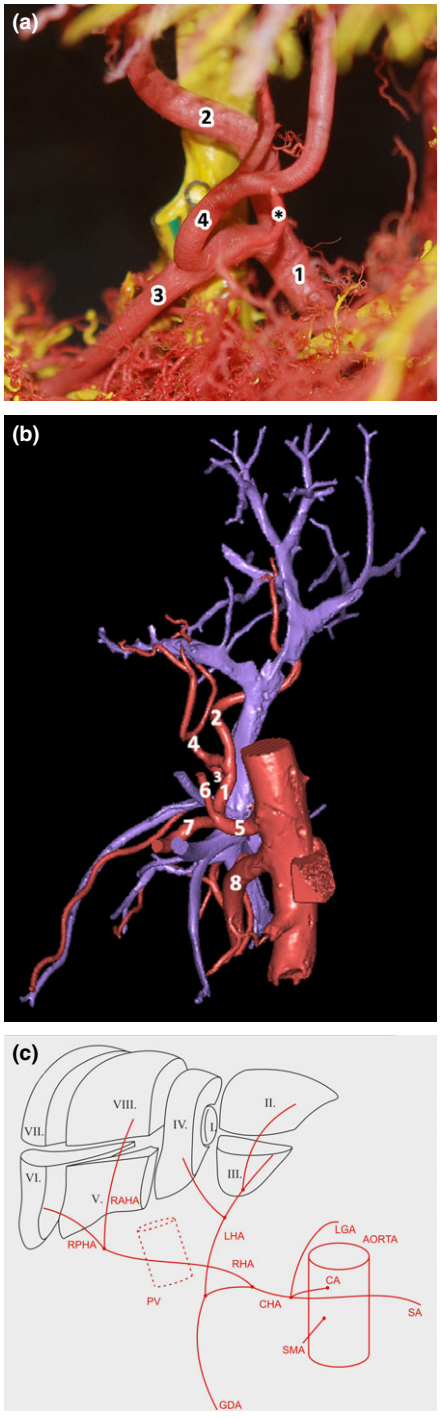

Figure 5 (a–c) Proximal branching of RHA, passing in front of the portal vein. New UC variant. 1: CHA; 2: RHA; 3: GDA; 4: LHA; 5: CA; 6: LGA; 7: SA; 8: SMA; *: RGA; PV: portal vein. (a) anterior aspect. (b) left lateral aspect.

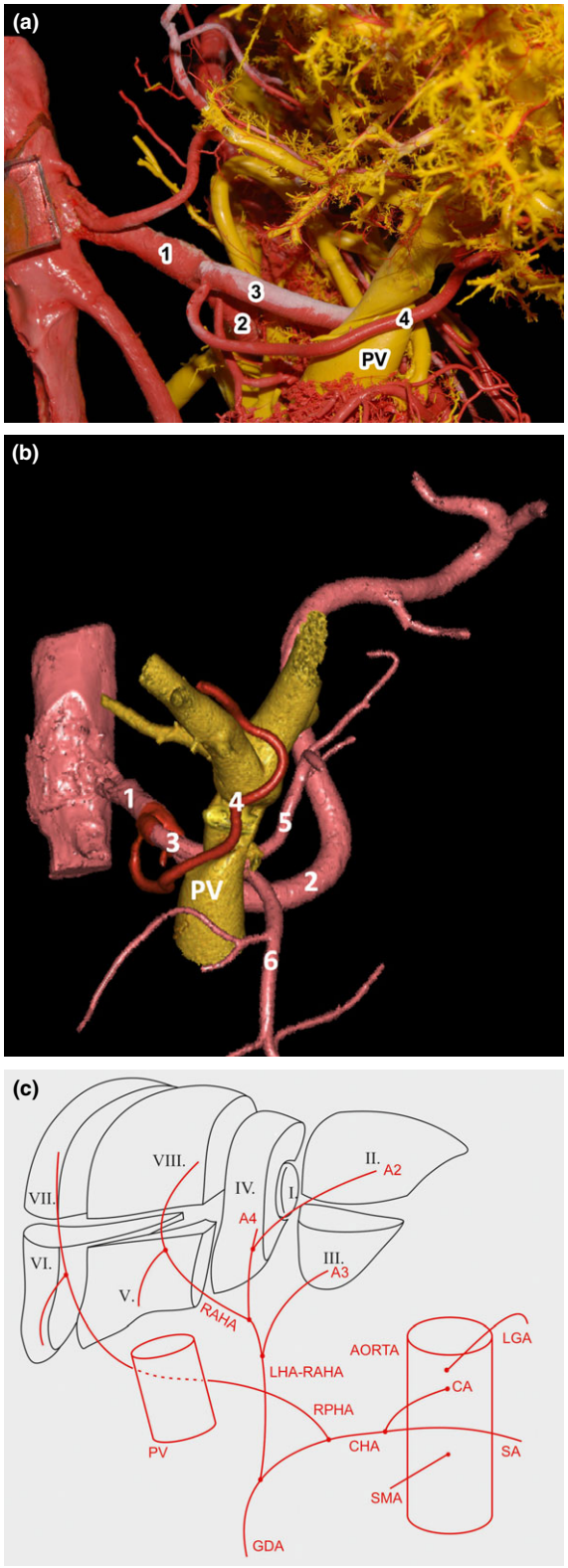

Figure 6 (a–c) Proximal branching of RPHA with retroportal course. New UC variant. 1: CA; 2: SA; 3: CHA; 4: RPHA; 5: LHA-RAHA common trunk; 6: GDA; PV: portal vein. (a,b) Right lateral aspect.

We would like to emphasize that vascular corrosion casting, when performed correctly, is an effective and reliable technique for clinical anatomical investigation of the

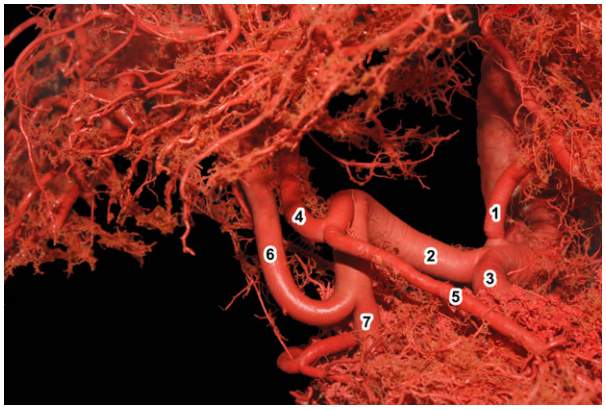

Figure 7 Proximal branching of LHA and RHA originating from GDA. 1: LGA; 2: CHA; 3: SA; 4: LHA; 5: RGA; 6: RHA; 7: GDA. Anterior aspect.

hepatic arterial system. The proper setting of the viscosity, the CT density and the color coding of the resin mixture developed by M. Kiss has the advantage of a highly detailed, real 3-dimensional demonstration of the hepatic arteries up to the 8th-order branching from the proper hepatic artery (PHA). This allows us to provide information about the complete abdominal vascular anatomy, including detection of new hepatic variations, identifying subvariants of previously reported cases and describing vascular structures that can be overlooked on CT angiographies or conventional angiograms. Lee *et al.* [8] reported that multidetector CT was unable to depict the origin of segment IV artery in 18% of liver donors, small accessory hepatic arteries in 13%, second-order branches of the left hepatic artery in 18%, second-order branches of the right hepatic artery in 6% due to technical limitations and respiratory motion artifacts. De Cecco *et al.* [9] and Koops *et al.* [10] share the opinion that the wrong positioning of the angiographic catheter, the small caliber and the slow flow in the aberrant hepatic arteries are the main reasons for problematic identification of these vessels. Therefore, high-resolution CT imaging and meticulous analysis of the images are strongly advised.

Our results show substantial differences concerning the variations of the extrahepatic arteries, compared to the literary data (Table 1). While, according to several authors, the incidence of the Michels I type ranges between 50.7% and 80.9% [1,2,9–14], we found normal anatomy in only 42% of cases. Surprisingly, the second most frequent variation in our study was the UC type with 18%. Only Coşkun *et al.* [12] reported high frequency of UC type (16.6%). Our series shares the general findings of the low percentage of Michels' types VII, VIII, and IX; however, discrepancies of other patterns are obvious. These may be explained with the low number of cases in our series, population differences, misinterpretation of radiological findings in other investigations due to respiratory motion artifacts, wrong catheter positioning, narrow diameter or slow flow in the small aberrant vessels. It is notable that unintentional wrong catheter positioning can be relatively common during radiological interventions and selective angiographies.

The limitation of our study is the relatively low number of cases; however, our results and other angiographic investigations in which the patient numbers are ranged between 40 and 63 [2,12,15,16] are comparable to the larger series as well [10,14,17,18,38,39].

In the 1990s, livers displaying aberrant or accessory right and left arteries and requiring multiple anastomoses were not frequently used [19,20]. As the routine use of arterial reconstruction techniques, such exclusions are extremely uncommon. However, the surgeons should take special care in identifying and distinguishing their size and position of these accessory and replaced arteries, based on preoperative high-resolution MDCT and/or MR angiographic data [21]. The surgical strategy depends largely on the diagnostic accuracy of the patient's vascular morphology. In contrast to the concepts of novel classifications (Hiatt *et al.* [5]; Abdullah *et al.* [18]), we share the opinion of Michels and recent clinical studies [17,22,23] making categorical distinction between accessory and replaced arteries. These authors point out that whereas replaced arteries must be always preserved, accessory vessels do not necessarily need to be reconstructed if intrahepatic anastomoses result in adequate back-flow or if intra-operative Doppler ultrasonography confirms sufficient perfusion of every liver segment [18]. The presence of accessory arteries, however, might necessitate reconstruction of multiple vessels which, due to their narrow diameter, leads to an increased risk of hepatic artery thrombosis. Consequently, not only the volume of the supplied liver parenchyma, but the length and caliber of these vessels are important factors in the planning, performance, and efficiency of arterial reconstruction.

Whereas some authors [15] state that small-diameter arteries included in Michels' classification are of no clinical relevance, others [2,9,13,24] point out that these vessels do affect the surgical planning and the placement of chemotherapy pump or embolization catheter in patients subjected to primary or metastatic liver tumor treatment. The reason is that variant anatomy may be the cause of incomplete embolization of the tumor, incomplete perfusion of the liver or liver remnant and extrahepatic perfusion, which may result in vessel thrombosis, misperfusion of chemotherapeutic or radiotherapeutic agents [2] pancreatitis or gastroduodenal ulcerations [25,26].

Regarding the UC variants, Abdullah *et al.* [18] published the highest number, actually 19 types (in 50 cases) which could not fit into Michels' classification, in their series of 932 surgical dissections in liver transplantation. Covey *et al.* [13] published 17 types (in 45 cases of 600),

Table 1. Variability of variations compared to other authors. Main differences are shown in bold italic values.

	Michels' types										
Author (Year)	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.	UC
Our series (2015) (*n* = 50) corrosion casts	***42***	6.0	14.0	4.0	8.0	0	2.0	4.0	2.0	0	***18***
Kamel *et al.* (2001) [15] (*n* = 40) MDCTA	70.0	5.0	7.5	2.5	7.5	2.5	0	5	0	0	0
Coşkun *et al.* (2005) [12] (*n* = 48) 16-row CTA	54.1	0	6.3	0	16.6	2.1	4.2	0	0	0	16.6
Ferrari *et al.* (2007) [16] (*n* = 60) 64-row CTA	60.0	10.0	18.3	5.0	1.7	0	0	1.7	0	0	3.3
Stemmler *et al.* (2004) [2] (*n* = 63) 4/8-row CTA	80.9	0	6.3	0	7.9	0	1.6	1.6	1.6	0	0
Varotti *et al.* (2004) [6] (*n* = 96) liver graft	70.8	6.25	10.4	2.1	6.25	3.1	0	0	1.1	0	0
Ugurel *et al.* (2010) [27] (*n* = 100) 16-row MDCT	52.0	11.0	17.0	1.0	10.0	1.0	1.0	1.0	2.0	0	4.0
De Santis *et al.* (2000) [39] (*n* = 150) angiography	52.0	10.0	15.5	0.6	0.6	2.0	0.6	0	4.0	0	14.7
Michels (1966) [4] (*n* = 200) cadaver dissection	55.0	10.0	11.0	1.0	8.0	7.0	1.0	2.0	2.5	0.5	0
Rygaard *et al.* (1986) [28] (*n* = 216) arteriographies	75.5	4.6	13.4	0.9	0	0	0.5	0.5	1.4	0	3.2
Kishi (2004) *et al.* [38] (*n* = 223) angiography	61.0	14.0	4.0	0	12.0	3.0	2.0	0	6.0	0	0
De Cecco *et al.* (2009) [9] (*n* = 250) 64-row CTA	66.0	5.2	9.2	2.0	5.2	4.0	2.0	0.6	2.0	0	3.3
Kishi (2010) *et al.* [11] (*n* = 361) angiography + CTA	68.6	10.2	6.9	4.2	4.7	1.4	0.6	0.6	2.5	0	0.3
Winston *et al.* (2007) [1] (*n* = 371) 4-row CTA	50.7	14.5	8.1	0	3.5	0	0	0	1.6	0	12.5
Covey *et al.* (2002) [13] (*n* = 600) DSA	61.3	3.8	8.7	0.5	10.7	1.5	1.0	3.0	2.0	0	7.5
Koops *et al.* (2004) [10] (*n* = 604) DSA	79.1	2.5	8.6	1.0	0.5	3.3	0.2	0.2	2.8	0	1.8
López-Andújar *et al.* (2007) [17] (*n* = 1081) liver graft	70.0	9.7	7.8	3.1	3.9	0.6	0.6	0.3	2.5	0	1.0
Saba and Mallarini (2011) [14] (*n* = 1629) MDCTA	61.37	7.48	10.56	1.35	6.69	6.99	0.73	1.9	1.59	0.31	1.09

Table 2. Unclassified variations published by other authors.

AUTHOR	UC variations	AUTHOR	UC variations
New variations in our series	CHA pentafurcation proximal RHA from CHA with anteportal course proximal RPHA from CHA with retroportal course prox., retroportal RHA + CHA trifurcation: LHA, GDA, RGA	**Lee SS**	RHA from CA ***CHA trifurcation*** celiacomesenteric trunk hepaticomesenteric trunk a-RHA from GDA or DPA ***proximal branching of LHA from CHA*** separate origins of S II. and S III. branches
Ferrari	CHA from aorta r-LHA from IPD + r-RHA from SMA		
De Cecco	r-RHA / r-LHA directly from aorta a-/r- RHA or LHA from CA, IPD, Bühler-arch	Winston	LHA from CA RHA from CA LHA from CHA RHA from GDA GDA from RHA RHA from aorta CHA from aorta GDA from SMA CHA trifurcation S IV. branch from GDA
Coskun	a-RHA from CA GDA from RHA ***CHA trifurcation*** a-RHA from CHA a-RHA from SMA + a-LHA from GDA		
Koops	r-LHA from GDA a-RHA from CA r-RHA from aorta r-LHA from GDA + r-RHA from SMA RHA + LHA separately from CA + GDA from LHA RHA + LHA separately from CA + GDA from RHA	**Braun**	r-RHA from right renal artery
		Wadhwa	retroportal course of PHA
Johnson	Celiacomesenteric trunk + proximal branching of LHA from CHA + LGA from aorta	**Nakamura**	a-LGA from LHA
		Polguj	a-RHA from GDA
Chaib	LHA from aorta or SMA	**Saba**	CHA from aorta r-CHA from SMA + a-LHA from LGA
Covey	CHA from aorta ***CHA trifurcation*** a-LHA from RAHA CHA quadrifurcation GDA from RHA or LHA RHA and/or LHA from CA a-RHA + a-LHA from LGA RHA and/or LHA from aorta r-RHA from right phrenic artery r-PHA from SMA + GDA from CA a-RHA from right phrenic artery or GDA or CA or LGA	**Lopez-Andujar**	CHA from aorta r-CHA from SMA + a-LHA from LGA
		Fasel	***RHA from CA with retroportal course***
		Gordon	a-RHA from dorsal pancreatic artery
		Ugurel	CHA from aorta RHA from aorta RHA from middle colic artery LHA from CHA
		Abdullah	CHA from aorta ***CHA trifurcation*** CHA with variations of GDA PHA with more than 2 branches a-/r-LHA from CA and/or a-/r-RHA from CA r-LHA from CA + r-RHA from SMA r-LHA + r-RHA from CHA r-RHA from IMA CHA but LHA gives PHA and RHA gives GDA retroportal CHA CHA + r-LHA + r-RHA (LGA from aorta) CHA from aorta + r-LHA CHA + r-LHA + r-RHA (CA and SMA origins at the same level) CHA + r-LHA (LGA from aorta) CHA (GDA from RHA) + a-RHA CHA (gives LHA + A4) + r-RHA CHA trifurcation (RHA, LHA, GDA) + a-RHA CHA trifurcation (RHA, LHA, GDA) + a-LHA CHA (gives GDA, RGA, LHA, RHA) + a-LHA + a-RHA
Soin	CHA from aorta a-/r-LHA from CA a-/r-RHA from CA a-LHA + a-RHA from CA a-/r-LHA from supracoeliac aorta a-LHA from CA + r-RHA from SMA a-/r-LHA from LGA arising from aorta a-/r-LHA from GDA + a-/r-RHA from SMA dual origin of single CHA from SMA and CA a-/r-RHA and CHA from SMA + a-/r-LHA from LGA		
Rygaard	RHA from aorta a-RHA from GDA double LHA from CHA r-LHA from LGA + LGA from aorta RHA from aorta + r-LHA from LGA RHA and LHA arise separately from CA r-RHA from SMA + a-RHA from GDA + r-LHA from LGA + LGA from aorta		

followed by Winston [1] with 10 NC types (11 cases of 50). Kishi *et al.* [11] report about r-RHA from dorsal pancreatic artery, r-LHA from CA, accessory S VI arteries (a-RHA) from PHA, CA, and superior posterior pancreaticoduodenal artery, combination of Michels V and IX; however, they only considered the latter case as UC variation. Table 2 shows a summary of extrahepatic variations not classified by Michels, observed in the current study and those described by other authors. Bold-italic letters show UC variations found also by us [1,8–10,12–14,16–19,27–36].

To the best of our knowledge, this study is the first to demonstrate four previously undescribed extrahepatic hilar arterial variants, which are to be recognized accurately before surgery in order to avoid graft injury and ensure a safe hepatectomy. These newly presented variants are as follows: (i) CHA pentafurcation; (ii) CHA trifurcation into LHA, RGA, and GDA together with a proximally originating, retroportal RHA; (iii) proximal branching and anteportal course of RHA from CHA; and (iv) RPHA deriving from CHA and traveling a retroportal course.

Pentafurcation of the CHA can be beneficial, if the right lobe, segment IV or segments II and III are involved in tumorous transformation. The sufficient length, large diameter, and easy identification of the RHA, LHA, and A4 – as seen on our preparation – allows the surgeon to safely perform right hepatectomy, left lateral split, or Taj Mahal resection [37], without compromising the arterialization of the liver remnant. This new arterial variation may not necessarily cause problems during superselective chemoembolizations; however, when whole-organ chemo- or radioembolization is needed, this anomaly can potentially lead to significant gastrointestinal side effects by shunting the therapeutic agents to the nonhepatic arteries. While this anatomic variant is manageable during whole-organ recovery from a deceased donor, it may be a problem potentially for a full left lobe donation.

Particular attention must be paid when the RHA or the RPHA displays a proximal origin from the CHA (or CA). After passing behind or in front of the portal vein, the distal part of the vessel reaches the right side of the hepatoduodenal ligament. Therefore, one has to be careful during dissection not to inflict accidental damage to the common bile duct, which runs close to it in the hepatic pedicle. In case of proximal branching of the RHA, the point of origin usually lies deep, next to the CA division, consequently – if liver volumetry allows – the left lobe is more preferable for donation in living donor liver transplantation due to its easier accessibility. However, in this case left lateral splitting may endanger the blood supply of segment IV, causing ischemia.

In case of the proximal branching of the RPHA, the right anterior sector of the liver is supplied by the common trunk of the LHA and RAHA and the first branch of this trunk is A3 on our cast. Subsequently, the intrahepatic distribution of the segmental arteries (A3 from LHA-RAHA trunk; A2 from A4) would result in two arterial stumps during left lateral splitting and the arterial inflow of S V and S VIII could also be endangered. Furthermore, a separate RPHA arising from CHA may imply a relative contraindication for right lobe living donation and full left – full right split as well, due to the double source of arterial supply of the right lobe (S V and S VIII from LHA; S VI and S VII from CHA). On the other hand, the proximal origin of the RHA or RPHA may have the advantage of an easier selective catheterization and a reduced risk of chemo- or radiotherapeutic agents reaching the wrong liver lobe.

Conclusion

Our method, simulating the MDCT angiography allows us to provide accurate information about the complete upper abdominal vascular anatomy, including detection of new hepatic arterial variations, identifying subvariants of previously described cases. Given the fact that all four new unclassified variations were accurately visualized on our 3D CT reconstructions, these structures should be identifiable during clinical CT examinations as well. Being of great surgical and radiological importance, unusual variations must always be in the focus of surgeons and radiologists during the preoperative evaluations and interventions in the upper abdomen.

We emphasize that comprehensive knowledge of the arterial variations and their prevalence is of crucial importance for visceral surgeons, who often have a limited opportunity of direct visualization of the surgical field because of the obesity of the patient, hepatobiliary and pancreatic malignancies or extensive local inflammation. Besides, the increasing number of living donor and deceased donor liver transplantations, anatomical and nonanatomical hepatic resections, laparoscopic, and radiological interventions are the main reasons for a renewed interest in the investigation and analysis of arterial variations and reports of new variants. The hepatic arterial anatomy is highly variable and some variations need careful preoperative planning of vascular reconstruction. We believe that our data may contribute to the better knowledge of the extrahepatic arterial supply of the liver and therefore lead to the reduction in perioperative and postprocedure complications.

Authorship

AS: performed the corrosion casts and their photo-documentation. MK and ÁN: worked in the method of making corrosion casts. CK: performed CT scans and reconstructions. RD, ZM and LK: discussed the clinical relevancies. KN: performed the corrosion casts and their photo-docu-

mentation, collected and reviewed the related publications, contributed in writing the study and reviewed by all authors.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Video S1. Video (proxRPHA.wmv): Rotating image of a new UC variation shown on Fig. 6a–c. Orange: aorta, CA, SA, SMA. Red: CHA, LHA-RAHA trunk, GDA; Yellow: proximal RPHA; Blue: PV.

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